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CURRENTS IN EXPERIMENTAL CONDITIONS

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by V. K. Boyko, P. F. Demidyuk,
A. A. Moybenko and G. A. Sidorenko

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DEFIBRILLATION OF THE HEART WITH LOW TENSION CURRENTS IN EXPERIMENTAL CONDITIONS

[This is a translation of an article written by Senior Scientific Worker V. K. Boyko, and scientific associates P. F. Demikyuk, A. A. Moybenko and G. A. Sidorenko in Novyy Khirurgicheskiy Arkhiv (New Surgical Archives), No 6, Kiev, 1959, pages 13-17.]

Department of Clinical Surgery (head-docent A. L. Pkhakadze) and department of Pathological Physiology (director-candidate of medical sciences A. I. Khomazyuk) of the Ukrainian Scientific Research Institute of Clinical Medicine.

Fibrillation represents one of the most serious disorders of cardiac activity and is manifested in variously timed, disorderly, shallow twitchings of individual fibres of the cardiac muscle. These chaotic contractions of the myocardium do not cease on their own accord in man and in large warm-blooded animals, and lead to death because of the disorder of the pumping function of the hearts.

Fibrillation occurs in mechanical trauma of the heart, including operative and electro-trauma, loss of blood, mechanical asphyxia, chloroform anesthesia, and drowning. This condition is observed in approximately five percent of cases in operations upon the heart in man, still more frequently in hypothermia.

Despite the fact that fibrillation has been known for a long time (more than 100 years) the mechanism of its origin is not yet fully clarified. The theory of nonadoption of a very frequent rhythm of stimulation by the most labile elements of the neuro-conductive pathways of the heart appears to be the most likely one. These impulses, having at first produced an extrasystole, when transmitted along the more labile elements, lead to an increase of the frequency of their (of the impulses) circular motion and, consequently, to a complete disturbance of coordination of the cardiac contractions. The continuation of the chaotic contractions of the individual fibres of the myocardium in man and in warm-blooded animals is explained by a particular structure of their heart, particularly of the so-called intercalated disks which play an inhibiting role in the transmission of the impulses. These, having met an obstacle on their way, accomplish a return movement, and thus call forth fortuitous but uninterrupted contractions of those adjacent heart elements, in which excitability was already restored (N. L. Gurvich).

Attempts of arrest of cardiac fibrillation were first undertaken in 1850 by Hoff and Ludwig. However these were rarely achieved with success. F. A. Andreyev arrested in 1933 fibrillation of the heart in a dog stricken by an electrical current with an injection into the carotid artery of a five percent solution of potassium chloride. This drug was also tested by Hooker and Shcherbakova. However, it was demonstrated subsequently that even though potassium chloride arrested cardiac fibrillation, the heart failed to resume its activity because of the onset of paralytic diastole. When a solution of potassium chloride was introduced into the heart under this condition, secondary fibrillation occurred. Therefore other pharmaceutical drugs were subsequently introduced such as: novocain, procaine, quinidine, papaverin, prostigmine Tareyev's mixture, etc.

All the measures used to combat fibrillation of the heart may be divided into the three following groups: by means of chemical, physical and biological nature. The above mentioned preparations and also adrenalin, refer to the first group. This last one, indeed, may by itself produce fibrillation in the absence of cardiac contractions.

Measures of physical order: massage and electrical current; biological: intraarterial blood transfusions, particularly into the coronary vessels.

It became clear with the accumulation of subsequent observations that chemical preparations in themselves are not important for defibrillation, and that they may be applied as auxilliary means facilitating the arrest of fibrillation by electrical current.

Indeed, not once did we succeed in our experiments (8) in the arrest of fibrillation by administration of only novocain, adrenalin, Tareyev's mixture, potassium chloride without simultaneous administration of electrical current. Only in experiment No 19 was the arrhythmia, which resulted following the section of the venae cavae arrested by the injection of potassium chloride into the left ventricle of the heart. Massage of the heart arrests fibrillation only in small warm-blooded animals (cats and rabbits) but it is also ineffective without the application of an electrical current in the dog and in man. We applied therefore, in our subsequent experiments, an electrical current of various intensity and tension as the most effective means of combatting fibrillation of the heart.

In the beginning we tried first and foremost to produce fibrillation by various operative interventions, particularly by comissurotomy, by inflicting a wound on the heart, followed by insertion of sutures, by anastomosis between the large vessels etc. We have succeeded in producing fibrillation of the heart in only one out of twenty-five cases (with comissurotomy), and that we explain by asphyxia and not by the trauma of the heart itself

since the dog, during the period of the operative intervention was breathing poorly, because of technical reasons, (for a certain period of time -- the apparatus for artificial respiration being defective) and found itself in a condition of severe asphyxia (experiment No 42). We have failed to produce fibrillation in experiments by inflicting a series of injuries to the heart, for instance, by its puncture, compression by blunt forceps, by punctures in the area of Shmey, section of the thoracic aorta and also of the vanea cavae. It is important to note that before the infliction of various orders of trauma in the described experiments we have irrigated the heart with a five percent solution of novocain and the pleural cavity with a one percent solution despite the fact that the animal was under intubation ether-carbon dioxide anesthesia and that an apparatus for artificial respiration was used. It is our opinion that these circumstances impeded the occurrence of fibrillation. Our assumption is confirmed by the opinion of A. N. Bakulev, who recommends thorough anesthesiation of the reflexogenic areas of the heart and the lungs for the prevention of fibrillation.

Having failed to produce fibrillation by mechanical trauma of the heart, we turned to a more reliable means -- the action of an electrical current either upon the whole organism or upon the heart only. We applied, at first, an electrical current from the illuminating circuit of 110 volts for a period of one second. However, this was followed only by arthmia of the heart. Later we began to apply a direct current of low tension -- 12 volts- and an alternating current of a higher tension- 110 volts, but for a comparatively longer period of time (up to five seconds).

Our method consisted of the following: under intubation ether-carbon dioxide, with the inclusion of the apparatus for artificial respiration, the chest cavity of the dog was opened by an incision in the 4th or 5th intercostal space on the left. Following irrigation of the pleura with a one percent solution of novocaine (from 40-60 ml) the pericardial cavity was injected with two-three ml five percent novocain. The pericardium was opened and two electrodes wrapped with moist gauze connected to the illuminating network or to an accumalator (battery) with a constant current of 12 volts and an intensity of 1.5-2 amperes were applied to the heart (upon the right auricale or/and upon the left ventricle). After passage of such a current through the heart for a period of three-five seconds, we invariable produced fibrillation. We accomplished this in isolated cases by passing an electrical current of low tension through the whole organism (the electrodes were applied to the right anterior and left posterior paw).

As we have mentioned, we have carried out defibrillation in the last series of experiments with the aid of an electrical

current for which purpose we have constructed a special defibrillator (Fig. 1). This consists of two metallic, somewhat bent rectangular electrodes measuring 4 x 4.5 cm which are connected with the aid of insulated wires into the illuminating circuit through a transformer and of a spring switch with a graduation of the time of inclusion of the electrical current (from 0.01 - 1 second and more). Immediately upon the appearance of fibrillation, but not later than within one-two minutes, we passed through the heart with the aid of the defibrillator, an electrical current of various tension and duration in the various experiments. We carried out a total of 60 such experiments. The following were the immediate results.

The electrical current of the illuminating circuit with a tension of 110 volts arrested fibrillation of the heart following a period of 0.1 seconds in two cases only. In one of those the heart action was restored fully following message and injection of adrenalin into the heart. The injection of adrenalin produced in the second case a secondary fibrillation which could not be arrested again, and the animal died.

In the following experiments, with defibrillation, we raised the tension of the electrical current to 240 volts, decreasing the duration of its action to 0.02 seconds. Having tried various combinations of duration of the action of the defibrillating electrical current, of the value of its tension and intensity, we adopted the following method.

By using this method we arrested fibrillation in the last series of experiments in all the cases (15 experiments). All the animals withstood the operative intervention safely and only two of them perished during the post-operative period due to incidental complications, not associated with the passage of the electrical current through the heart. It is noteworthy that in order to arrest fibrillation it is necessary to apply an electrical current of higher tension as compared with the current which produced the fibrillation. Thus, in experiment No 36, an intensity of the defibrillating current of 175 volts was needed in order to arrest fibrillation produced by a direct current of 12 volts. Fibrillation produced by an alternating current of the illuminating circuit of 110 volts was arrested only with an electrocurrent of 240 volts tension. Our methods of defrillation were so effective that in some cases fibrillation was arrested in one and the same animal two or three times following its artificial production (experiments Nos 46 and 48).

In one of the experiments fibrillation was stopped five times in one and the same animal of which four times -- following its spontaneous recurrence. We will take the liberty of describing this last experiment in more detail.

Experiment No 47. A male dog weighing 26 kg was subjected to a thoracotomy on 10 April 1958 by an incision in the fourth left

intercostal space while under intubation ether-carbon dioxide anesthesia and the application of the apparatus for artificial respiration. Following irrigation of the pleural cavity with 60 ml of one percent novocain and the injection into the cavity of the pericardium of three ml of five percent novocain, the pericardium was dissected. An electrical current from the illuminating circuit, with an intensity of 110 volts, was passed through the heart for a period of one second. Fibrillation appeared. Within 1.5 minutes a current of 240 volts tension and 1.5 ampers intensity, 0.22 seconds duration was passed three times at intervals of one second through the defibrillator constructed by us. Fibrillation was arrested. Weak isolated contractions of the myocardium appeared. 0.3 ml of 1:1000 adrenalin were injected into the left ventricle. Cardiac message. Following cessation of the message-recurrence of the fibrillation. Defibrillation was again carried out by the same method. Message of the heart caused a recurrence of its fibrillation. It was arrested for the third time by the same method and this was repeated five times. The last time, the fifth time besides the electrical current, a five-percent potassium chloride solution (2 ml) and 0.5 ml of adrenalin (1:1000) were injected into the left ventricle. Following this, the arterial pressure rose to the original level, and fibrillation did not recur although tachycardia appeared. The experiment was concluded successfully, the animal recovered.

Thus it appears that an electrical current of 240 volts acting upon the heart by brief impulses (of 0.02 second duration) in combination with message of the heart and injection into the heart of adrenalin, constitute a fully effective measure in combatting fibrillation. The described method may therefore be recommended for arresting fibrillation occurring during operation upon the heart.

Does the application of the described method for the purpose of defibrillation show any harmful effect upon the heart? L. D. Krymskiy and D. N. Tsukerman microscopically investigated the heart of the animals following electrical defibrillation. They found that the electrical current does not produce notable changes in the myocardium, but they observed, in the preparations following message of the heart, multiple hemorrhages in the thickness of the epicardium and the myocardium, and occasionally, edema of the last one. Our pathologo-histological investigations of the heart of dogs who perished following the experiment failed to demonstrate any important changes (A. I. Smirnova-Zamkova). We explain this by the fact that in our experiments message was not particularly prolonged - no longer than eight-ten minutes.

What is the mechanism of defibrillation with electrical Shock?

Defibrillation, under the action of electrical current (electroshock), was explained until now by a decrease of cardiac

excitability. Indeed, the basis of fibrillation consists in the hyper excitability of the myocardium; therefore, all the recommended chemopharmaceutical means against defibrillation were directed towards a decrease of the excitability of the heart. Numerous observations demonstrated that those drugs, in the majority of cases, not only arrested fibrillation, but also caused an arrest of cardiac activity. It is possible that this is related to the ignorance of a suitable effective and harmless dosage in their administration. As far as the action of the electrical current in defibrillation is concerned there is an accumulation of data at the present time showing evidence that it produces contraction of the ventricles following its repeated passage through the heart. N. L. Gurvich in his experiments for the purpose of elucidation of the mechanism of the action of the defibrillating current, arrived at the conclusion that the last one acts identically upon the fibrillation and normal heart. It is interesting to note that the cardiac activity was restored more rapidly with a 0.1 second duration of the current action than with an action of one second duration. We suggest that the electrical current, in its passage through the heart, acts in two ways: at first, it arrests, for a brief instant, cardiac contractions, including fibrillation, and then brings forth contraction of the ventricles. We have observed in our experiments, with passage of an electrical current through the heart for the purpose of producing fibrillation, that, in those cases, when the last one did not occur, a momentary fallout of the cardiac contraction, followed by their normalization, took place. It is to be supposed, therefore, that the defibrillating electrical current, having at first arrested fibrillation of the heart, brings forth consecutively a contraction of the myocardium and that by sending of such electrical impulses at intervals of 1-1.5 seconds, leads to the normalization of the rhythm of the cardiac contractions. However, these are quite weak during the first instances following defibrillation, and therefore, it is necessary to administer adrenalin and cardiac massage in order to fully restore cardiac activity.

Conclusions

1. Mechanical trauma of the heart causes fibrillation very rarely by itself, particularly when associated with preliminary irrigation with novocain.
2. Hypoxia of the myocardium appears to be one of the important causes of the occurrence of fibrillation.
3. Defibrillation by chemical drugs seems hopeless. It should be used in combination with other measures.
4. Defibrillation of the heart in dogs may be easily attained by the action of two-three impulses of an electrical current of 240

volts tension, 1.5-2 ampers intensity, 0.02 seconds duration, followed by massage and injection of adrenalin into the heart.

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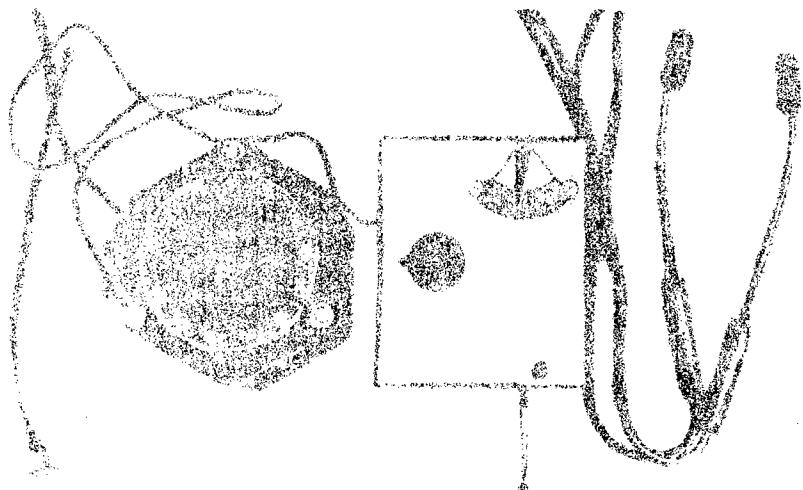


Fig. 1

Following the appearance of fibrillation an electrical current of 240 volts, and of an intensity of 1.5-2 amperes, of 0.02 seconds duration was passed through the heart. Two-three impulses at intervals of 1-1.5 seconds were usually needed in order to arrest fibrillation. The cardiac contractions, following the arrest of fibrillation, were weak. We have, therefore, immediately applied massage of the heart for a period of one-two minutes, following which we injected into the left ventricle 0.3-0.5 of adrenalin (1:1000 and again continued with massage. As a rule, within two-three minutes following the injection of adrenalin, the cardiac contractions became stronger, the arterial pressure increased considerably above the original level, decreasing, somewhat, at the end of the experiment. No recurrence of fibrillation took place.

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FOR REASONS OF SPEED AND ECONOMY
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